BACTERIAL ARTHRITIS

A Review

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Abstract: Acute pain in peripheral joints is not a common presenting symptom for chiropractors or osteopaths. However, chiropractors or osteopaths may be asked to assess peripheral joints when patients present with other conditions such as back pain.

This paper reviews the literature on bacterial arthritis as a specific type of infectious arthritis. Information was obtained from Medline and internet search using the keyword: "bacterial arthritis". The most common presenting symptoms are described, with specific reference for chiropractors and osteopaths in clinical presentation of patients' with this condition.

INTRODUCTION

Infectious arthritis is inflammation of a joint which has been caused by either bacterial, viral or fungal infection. Bacterial arthritis is defined as an arthritis resulting from infection of the synovial tissues with pyogenic bacteria or other infectious agents (1). In 95% of cases, acute infectious arthritis is caused by either bacteria or viruses.

Bacterial Arthritis (BA) can be categorized into two groups:

1. Arthritis due to Neisseria gonorrhoeae or other Neisseria species (the most common).
2. Non-gonococcal bacterial arthritis (Staphylococcus aureus being the most common bacteria) (2).

The epidemiology and clinical features of bacterial arthritis have changed recently. This can be attributed to factors such as longer life expectancy, increased frequency of methicillin-resistant (an antibiotic) Staphylococcus aureus isolates, increased use of arthroscopy, increased proportion of individuals with prosthetic joints and the spread of the AIDS epidemic (3).

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In general most cases of bacterial arthritis present with mono-articular joint pain and swelling, with fever being variable and occurring in 30 - 60% of patients (4).

Research into the incidence of bacterial arthritis in Western countries demonstrates that gonococcus remains the most common form of bacterial arthritis (5).

Pathogenesis and Pathology

Except in the case of direct penetration of the joint capsule via trauma or during operative procedures, the most common path of joint invasion by bacteria is the haematogenous route. This usually reflects the failure of the hosts systemic immune defence mechanisms (6).

Infection of joints via the infection of other organ systems may not often be clinically apparent. Other causes of infection of the joint may be a result of the course of other disease processes such as osteomyelitis, cellulitis, abscesses, tenosynovitis or septic bursitis (7-9).

Micro-organisms penetration of the joint via any route other than direct inoculation is an unlikely portal of entry. This is demonstrated by the fact that overt joint sepsis is of relatively low incidence when compared with the much higher incidence of systemic infectious diseases and bacteraemia (10).

The histopathological and biochemical changes observed in bacterial arthritis regardless of the route of infection are very similar (11,12).

The subsynovial space contains many polymorphonuclear leukocytes (PMNs). Phagocytosis of microorganisms by PMNs or by the synovial lining cells cause neovascularisation, synovial proliferation, granulation tissue and cartilage destruction (13).

PMNs then fill the joint cavity which gives synovial fluid its cloudy aspect as seen on aspiration. On examination synovial fluid contains decreased amounts of glucose and increased levels of lactate. This is expected in any active bacterial metabolism process. Also the synovial fluid will contain proteolytic enzymes, products of bone and cartilage degradation and pro-inflammatory substances.

Cartilage and bone destruction can be attributed to the interplay of:

The direct toxic effect of the microorganism involved,

Intense phagocytic activity of the synovial lining cells, PMNs and other cellular elements,
Proteolytic enzyme release,
Pro-inflammatory substances:
IL-1, TNF-a, CD-4 and other T-cells (13-15).

The amount of cartilage, bone and synovial degradation depends largely on the virulence of the invading organism, the host infection response, the promptness of diagnosis of bacterial arthritis, and subsequent treatment implementation (7,16,17).

The common agents that cause infectious arthritis include both cocci and bacilli. In a study by Fink and Nelson (14) the most common microorganisms found causing acute bacterial arthritis were:

Gram-positive cocci - Staphylococcus aureus (S. aureus), Streptococcus pyogenes, Streptococcus pneumonia, Streptococcus viridans
Gram-negative cocci - Neisseria gonorrhoeae and Neisseria meningitidis, Haemophilus influenza (This is a coccobacilli, often mistaken on smears as cocci).
Gram-negative bacilli - Escherichia coli, Salmonella, Pseudomonas.

Rutherburg and Ho found the incidence to be 17% of 412 cases reviewed in adults between 1934 and 1980, and that this as a cause of bacterial arthritis is steadily increasing in frequency (18).

S. aureus studies on animals have demonstrated the importance of various genetic and immunologic factors (19). Collagen binding protein adhering S. aureus to cartilage was found to be the "chromosomal collagen adhesion" gene or the CCA gene. Binding was found to have a direct correlation to this gene. Seventy percent of mice injected with activated CCA developed arthritis and only 27% of mice injected with deactivated CCA developed arthritis. Interleukon (IL-6) and immunoglubulin (IgG-1) levels were higher in the CCA activated group (20).

In another study, S. aureus injection intrevenously in mice showed increased amounts of granulocytes and macrophages in the synovium. The macrophages present included IL-6 and TNF-a. Forty eight hours after injection an infiltration of the T-lymphocytes CD-4+ ,IL-2 receptor and interferon were present (19).

Many synovial cells expressed major HLA class II molecules. Those rats effected had raised IL-6 and raised Polyclonal B cell activity. Results indicated T-lymphocytes contribute to an erosive and persistent course of S. aureus arthritis. Rheumatoid factor levels existed and remained elevated throughout the course of the disease (13).

Clinical Manifestations

Most non-gonococcal bacterial arthritis cases are mono-articular (up to 80% of cases). The knee is the most common joint effected (7). Patients present with constitutional symptoms including fever, chills and malaise, as well as evidence of onset of monoarticular and less frequently polyarticular involvement of joints (7,17,21).

An infant will usually present with irritability and fever. Examination will reveal failure to spontaneously move the limb involving the infected joint(s). Pain will be ilicited on passive movement of the limb (22). Acute pain, combined with stiffness will be the complaint of older children and adults. The joint is warm, tender and swollen on examination with signs of effusion (22).

In gonococcal infection, the arthritis will occur in conjunction with Disseminated Gonococcal Infection (DGI). DGI is known to occur in 1% of cases of gonorrhoea in the USA, and gonococcal arthritis has been demonstrated to occur at rates of 40 - 50% in various studies (14). The patient may present with or without migratory polyarthralgia, tenosynovitis, dermatitis, characteristic rash, fever and genitourinary symptoms. Usually multiple joints are involved, mostly the knees, wrists and ankles, although many other joints can be involved (14).

Clinically, the synovial fluid signs are similar to those in non-gonococcal bacterial arthritis. That is, synovial fluid count of 50,000 cells/mm3 , with the majority of patients being febrile, and having a modest peripheral leukocyte count (23). There are two basic types, a penicillin resistant (contains penicillinase) and penicillin sensitive (does not contain penicillinase). The penicillinase containing type commonly results in joint destruction. Generally there is a predominance of females to males 2-3:1, and patients are generally at a sexually active age (24).

Wise et al, found that only two of 41 cases of gonococcal bacterial arthritis were penicillinase positive (25). In contrast, in a South African study it was shown that 18 of 32 patients were penicillinase positive. These experiences demonstrated in a clinical setting for means of treatment, one must assume penicillin resistance until otherwise verified.

DIAGNOSIS

Diagnosis of BA involves the clinical picture of a red, swollen, tender joint and fever (17,53). The joints most commonly affected are the knee, hip, wrist and shoulder. Joint effusion is also expected, and if effusion is not present, a non-infectious arthropathy can be diagnosed (17). A joint effusion can usually be demonstrated in 90% of joint(s) affected with bacteria (14).

Identification of the organism involved is necessary for diagnosis and effective management. Culture of the synovial fluid (SF) may isolate the organism in 60% of cases. When SF cultures can't identify the organism, blood cultures may be useful in another 14% of cases. Failing this, local sites of infection must be cultured. The culturing of involved sites is especially important in cases involving gonococcal infection as this infection is mostly triggered by non-infective immune mechanisms (38,54).

The single most important investigation is the demonstration of SF leukocytosis and bacteria on the gram stain. A leukcytosis of less that 20,000x106/l makes infection unlikely, but does not exclude it, especially where gonococcus is concerned (53). Other authors list the critical range of total leukocyte count between 50,000-20,000 cells/ml (50,000-200,000x106/L). PMNs are expected to be present in approximately 90% of cases, but are generally present in over 75% (23).

Fink and Johnson state that in the diagnosis of non-gonococcal BA, the gram stain is positive in 75% of cases for gram positive cocci (2). Cell counts of greater than 50,000 leukocytes/ml, occur in 70% of patients, and over 80% demonstrate the presence of neutrophils. Synovial fluid glucose concentration should be much lower than serum glucose, with blood cultures positive in 50% of non-gonococcal cases and an elevated white cell count. An elevated ESR is often present, however, this is not specific to BA.

Notably SF lactic acid count was determined to be not diagnostic between BA and other forms of inflammatory athritis (53,55). Anaemia is unlikely to present early unless another disease associated with anaemia already exists (18).

Various studies have relied of different diagnostic criteria. A study by Unkita-Kallio et al, used the diagnostic utility of C-reactive protein, ESR, peripheral blood leukocyte count, and these were compared between patients with septic (or bacterial) arthritis plus osteomyelitis and patient with osteomyelitis alone (56). C-reactive protein was higher and remained higher in the combined group compared to the osteomyelitis alone group.

Clinical studies using scintillography to diagnose joint and bone infection have been performed. The results of these studies include:

1. Indium III - IgG - imaging for comparable infections with Indium labeled leukocytes and Technetium 99m immunoglobulin (57).

2. Tason et al, testing isotope bone scan accuracy in a study involving 86 children, revealed accuracy to be 81% (58). Soft tissue infection, adjacent to the joint involved, gave some false positive results.

A study by Sandrasegaran using MRI, finding it useful in the imaging of the SI joint in cases of septic sacroiliitis (59).

The following are a list of diagnostic tests (apart from clinical/laboratory testing procedures) which are often performed in the diagnosis of BA (18).

Radiological Examination

Several weeks are required before visible signs of infections are evident in bone and cartilage seen on the radiograph. Rarefaction of subchondral bone occurs first, followed by erosion of juxta-articular bone and erosion of the joint space following the destruction of the articular cartilage. In the joint that is already damaged (eg. by a prior arthritic condition), changes due to BA may not be evident. In the deep joints (eg. the hip, SI joints) soft tissue changes as a result of distension of the joint may also be seen.

Arthrography

Arthrography is useful for imaging capsular or ligamentous damage. The synovial fluid obtained should be examined for evidence of microorganisms by staining and culture before contrast dye is injected (18).

Computed Tomography (CT)

Computed Tomography is less useful in examining the peripheral joints and neck. It is good for examining deep-seated areas of swelling and effusion, such as in SI joint involvement.

Magnetic resonance imaging (MRI)

MRI is important for imagining both bone and soft tissue. Therefore, extension of infection to adjacent soft tissue can be delineated. Whilst MRI can demonstrate joint involvement, X-ray and CT scan are also both capable, although less sensitive in making a quick and accurate diagnosis. MRI has been found to be very useful in viewing the SI joint (5).

Bone Scan - Radioisotopic Scintography

99m diphosphonate + 67 Gallium citrate scintography may detect infection at an early stage of involvement. This is very useful in examining the small deep joints of the spine, including the facets and other joints.

It is important to note that whilst bone scans, CT and MRI can detect infection at an early stage, positive findings for all of these tests are not specific for infection. Other inflammatory diseases can produce similar findings to those of BA. The complement of the symptoms from the

patient history, imaging, and blood testing, can provide the most likely differentiation between infection or inflammation in joints.

Bacterial arthritis in the Child

The age of a child is the most important determinant of susceptibility of the host to a specific pathogen. Also important is the environment in which the pathogen is acquired, either community or nosocominally (hospital) acquired (14). In the neonate aged under 6 months, S. aureus is the dominant pathogen. In children of age 5 and over this is also the case (14).

Ho noted with hospital acquired pathogens in the neonate, the relative organism rates were: Staphylococcus (62%), Candida (17%), gram negative bacilli (13%) Streptococcus and Haemophilus influenzae (4% each) (35). This is compared with neonate infection which was community acquired, where the causing pathogens were Group B Streptococcus (52%), Staphylococcus (25%), Gonococcus (17%) and gram negative bacilli (5%). Ho also noted that, whilst this may not be a true representation of all or indeed any regions of bacterial infection, it does display the fact that a local predisposition to various pathogens exists depending on the environment in which it was acquired (35).

It should be noted that in the neonate that presents as very sick, joint involvement may be limited to one joint (21,26,27). When this is the case, bone and soft tissue may often become involved (26-28). It has also been found that polymicrobial infection may often be involved in the very sick infant (3).

In children over 6 months of age with bacterial arthritis and less than 2 years, Hemophilous influenzae and S. aureus are the most common pathogens (14). The ankle and hip, then to a lesser extent the knee are involved (29). The hip as a site of infection is difficult to diagnose, and in the child, failure to weight bear or pain on doing so should be considered as bacterial arthritis (3). Care should be exercised by the practitioner in excluding transient synovitis presenting primarily as mid knee pain in the child (3). In these children, infection in 80% of cases involves the lower extremity. The typical presentation is characterised by fever associated with focal joint findings. Identification of an etiologic agent from cultures of blood or synovial fluid can be expected in over 60% of cases. In up to 40% of cases, the joint fluid can be sterile (18).

Bacterial arthritis in Adults

In the adult the most common form of bacterial arthritis is caused by Neisseria gonnorrhoea (3,24). Of the non-gonococcal type, infections due to Gram positive organisms, especially S. aureus are the most commonly encountered articular infection within this age group (3,7,24). The frequency of Methicillin-resistant S. aureus within this age group has in recent years shown a marked rise (3). In reviews made by Mikhail and Alarcon (3), of the coagulase-negative staphylococci, Staphylococcus epidermidis remains a common pathogen, especially in prosthetic joints. Infection as a result of Gram negative microorganisms often occurs in the presence of pre-existing factors. The knee is the most common joint involved in the adult (5).

Bacterial Arthritis in the Elderly

Bacterial arthritis in the elderly differs from that in the adult and younger age groups. The differences include the prevalence of predisposing factors, variation in clinical features, causative organisms, delay in diagnosis and poorer outcome. Predisposing factors include an underlying arthropathy in 71% of individuals and clinical features displaying a high proportion of hip infection (38%). In the elderly, constitutional features displaying a diminished toxic state have also been shown, therefore delaying diagnosis and subsequently leading to poorer prognosis (30).

A high prevalence of underlying joint disease was found in 71% of sufferers in a study by Cooper et al (31). Infection commonly involved the hip (38%) and showed a poorer outcome than in the child or adult, were all noted in this study of 21 elderly patients in an English health district between 1973 and 1982 (31). Another English study, found that the number of the elderly people in Britain (over 60 y.o.) has increased, and the incidence of bacterial arthritis has also increased proportionally (32). Predisposing factors such as pre-existing joint disease, minor trauma, immunosuppression, diabetes mellitus, cirrhosis, chronic renal failure, rheumatoid arthritis and neoplastic disease have been noted (31).

The most common causing pathogen in the elderly is S. aureus with a frequency of 43 to 63 % of cases (31,33-36). The likelihood of a Gram negative bacilli as the cause of bacterial arthritis in the elderly is 24%. This is lower than the predilection of Gram negative bacilli as the primary cause of bacterial arthritis in the average adult population due to these pathogens infecting primarily intravenous drug users. It has been shown that the prevalence of this activity in elderly people is considerably less than that for younger generations. The simultaneous occurrence of septic arthritis and crystal induced arthritis appears to be a problem unique to the elderly (33).

Bacterial arthritis associated with chronic rheumatic disorders

When a patient has a longstanding, erosive, destructive seropositive case of rhematoid arthritis, there is an

increased likelihood of bacterial arthritis than in a patient with a less severe disease. (37,38). The fact that a joint with rheumatoid arthritis is prone to bacterial arthritis was first noted in 1958 (30).

Predisposing factors to bacterial arthritis:

Overall health status,
Comorbidities such as Diabetes Mellitis,
Administration of corticosteroids or cytotoxic drugs to suppress immunity,
Intra-articular use of corticosteroids (39).

Generally a bacterial arthritis involved rheumatoid joint will be more painful, tender and swollen than other rheumatoid arthritis joints, although this distinction between joints is difficult to determine (40,41). The large majority of bacterial arthritis in the rheumatoid arthritis patient will occur in a single joint. The knee is the most common joint, and S. aureus is the most common pathogen involved (3,30).

Typically patients with rheumatoid arthritis that contract bacterial arthritis are older. Often symptons are blunted by the patients receiving systemic corticosteroids. It was found in a review by Ho that just over half of cases have peripheral blood leukocytosis (35). Of infections involving gram positive cocci 89% were S. aureus which was the most common. Gram negative bacilli were responsible for the rest of the infections. Poly-articular infection occurred in 25% of cases, large joints especially the knee being the most effected (14).

Whilst it is known that rheumatoid arthritis is a determinant of bacterial arthritis incidence, it is unclear if crystal induced arthropathies or osteoarthritis are involved in bacterial arthritis (42,43). Patients with systemic connective tissue diseases may develop bacterial arthritis due to infection that would less likely cause bacterial arthritis in healthy adults, especially in Systemic Lupus Erythematosus (SLE) (7,38).

Bacterial arthritis involvement with Prosthetic Joints

If infection does occur with prosthetic joints, it will occur most likely in the early post-operative period (44,45). Early post-operative infection is caused generally by S. epidermidis or anaerobes, late post-operative infection is caused by gram positive or gram negative organisms (44,45). There is a necessity for prevention of these infections during both of these periods (3).

Often removal of the prosthesis will halt the progression to chronic inflammation (45). Infection rates have decreased due to recent improvements in surgical procedures. The use of antibiotics during operations is desirable in procedures where bacteraemia is a likely occurrence. This is particularly significant following dental and urological procedures (37,46).

Factors that predispose bacterial arthritis in patients with joint prosthesis include:

Impaired post defence,
Surgical technique employed,
Duration of operation,
Material and design of prosthesis, and Rheumatoid arthritis.

In optimal conditions, the overall infection rate in total joint replacement is close to 1% for the hip, and 2% for the knee. However, in a review McCarty and Koopman (1993), the involvement in the knee was as high as 4% of all cases (14).

Early infection in patients with joint prosthesis, often begins with post-operative wound infection. These infections respond to prompt and aggressive treatment and the original prosthesis can often be retained. Late infection usually results from haematogenous dissemination of bacteria. These late infections are more common in cases of individuals also suffering from rheumatoid arthritis (14).

According to Brause, pain accompanies infection 95% of the time (45). Fever occurs 43% of the time and swelling 38%. Drainage through the skin occurs 32% of the time. The possibility of infection should always be entertained when:

A prosthesis becomes painful,
Local signs of inflammation and drainage appear,
There are progressive signs of loosening of the prosthesis on x-ray.

Infection most commonly is caused by gram positive cocci, 75 - 80% of the time. Aerobic gram negative rods 10 - 20% of the time and anaerobic micro-organisms 5 - 10%. Staphylococci account for 75 - 90% of the gram positive cocci infections. S. epidermis occurs more regularly than S. aureus (45).

According to Mikhail, arthroscopy of joints does not pose the same infection risks as full joint replacements (3). This can be considered due to the decreased invasiveness and shorter duration of an arthroscopy. Infections that do occur involve mostly skin pathogens eg. S. epidermis, and are very difficult to irradicate.

Bacterial arthritis in the Immunocompromised Host

As previously stated, those people in the community that are in an immuno-compromised state are at a greater risk than others in the community of developing bacterial arthritis. These include:

The very young and the very old with multiple organ system involvement (4,47),

Those with HIV (Human Immunodefficiency Virus) (48,49),
Those that receive organ transplants (50), and
Immunosuppressant drug use in non-malignant disorders (51).

When the individual is immunocompromised polymicrobial and polyarticular arthritis may occur. Polyarticular arthritis will occur in 5 - 8% of paediatric cases and 10 - 19% of non-gonococcal adult cases (14).

Certain bacteria have a predelection for being polyarticular, including N. gonorrhoeae, S. pneumoniae, group G streptococcus, H. influenzae but most commonly S. aureus. Often in polyarticular infection, prior disease will be a factor e.g. Rheumatoid arthritis. It is unlikely that polyarticular infection will occur in a natural joint unless penetrating trauma, or recent surgery is a factor (14). In various cases of polymicrobial infection, combinations of usually two microorganisms have been found (14).

A study by Hughes et al (49), was done on the incidence of bone, joint and muscle lesions associated with HIV. This study indicated musculoskeletal sepsis in 10 cases out of a possible 3000 individuals with HIV. The study also showed that septic arthritis had been identified in 14 patients with HIV infection. This would indicate that septic arthritis and osteomyelitis might be more prevalent than previously thought in HIV sufferers. S. aureus or Streptococcus pneumonia were implicated in the majority of cases. Whilst it was accepted that musculoskeletal sepsis is uncommon in HIV infected individuals, evidence has been presented that this prevalence is increasing as a predisposing factor for septic arthritis (49,52).

Differential Diagnosis

Differential diagnosis depends on age, in that there are few rheumatic disorders that exist in childhood, diagnosis of the septic arthritity, especially when the condition is monoarticular. If the condition is polyarticular, in the child or young adult the following conditions can be considered:

Acute rheumatic fever
Lyme disease
Kawasaki syndrome
Viral infections

(or rarely reactive arthritis which presents with positive synovial fluid cultures).

In older individuals after ascertaining whether predisposing factors exist or whether a pathological process is present, crystal induced arthritis should be considered. Differentiation can be made by examination of synovial fluid under microscope, although the presence of crystals does not rule out bacterial arthritis. Psuedoseptic arthritis, infectious arthritis and reactive arthritis can all also be considered (3).

In a diagnosis of bacterial arthritis in the elderly, a prior history of crystal induced arthropathy can assist in the diagnosis of BA. Other diseases to be considered include Palandromic Rheumatism, the Spondyloarthropathies and Stills Disease (3).

Jones et al (1990), in a study on septic arthritis and its complications with appetite associated destructive arthropathy (AADA) showed there is a shared similar onset (6). This includes an initial, rapidly progressive, severely painful course demonstrating radiographs with rapidly destructive changes of bone and cartilage. Radiographic features differentiating AADA from septic arthritis include absence of osteopenic erosions and periostitis. These changes manifest within several weeks in the case of septic arthritis (6).

Treatment

The aim of treatment is eliminate infection, primarily through the use of antibiotic therapy. Four basic regimes exist in the treatment of bacterial arthritis:

Antibiotics
Drainage
Joint immobilisation and mobilisation (3)

Antibiotics

Antibiotics should be used when bacterial arthritis is first suspected, yet not proven by gram stain. The antibiotic originally used should vary depending on the way infection was acquired (3). If a patient should present as very sick with multiple joint infection, antibiotic treatment focusing for both gram positive and negative organisms should be given until the organism is identified (7,21).

For normal S. aureus infection the antibiotics that can be administered include numerous types such as methacycline (brand name- Rondomycin), ciproflaxacin (brand name- Ciproxin) and refampin. In cases of infection by strains of methycillin - resistant S. aureus, Vancomycin should be administered. When the microorganism is gram negative, a first or third generation cephalosporin or aminoglycoside such as Amikacin or Tobramycin. These are preferable to Gentamicin, to which most gram-negative organisms have an aquired resistence (1,14).

If in the case of a microorganism not being identified yet the initial antibiotic treatment is effective, the course of treatment should be continued. Once the antibiotic sensitivity of the organism has been determined, treatment can be provided on this basis (14).

Treatment Regime

The parenteral route should be maintained for no less than 4 weeks. This may be shorter if followed by the oral route with good result. Adequate bacteriacidal levels and good compliance are necessary for successful treatment. Long term administration of oral antibiotics is recommended in patients with protracted bone and joint infections, as is common with prosthetic joints. Following treatment in hospital, a Hickman catheter may be used for home administration of antibiotics (14).

It is known that antibiotics diffuse readily from the circulation into infected and non-infected joints (6). In a review by Schmid (1992), it has been shown that parental administration of various bacterial drugs have produced bactericidal fluid concentrations within the joint of effective values (17). This shows that there is no necessity to directly invade the joint space with antibiotics that can potentially create further infection. In such an occurence, chemical synovitis due to local irritation by the drug or needle can occur (39).

The general rule in using antibiotics to treat bacterial/ infectious arthritis is that most drugs reach therapeutic levels in the synovial fluid if adequate levels are administered systematically (17).

Drainage

Needle aspiration is recommended daily until either the synovial fluid aspired is sterile, or effusion is minimal. Joint lavage has been used in the past, yet this is no longer recommended. Joint lavage is common in patients deemed to be poor surgical candidates.

Surgical drainage is indicated in:

All cases of hip involvement.
Joints that don't respond to antibiotic therapy.
Joints where synovial fluid appears to be located.
Joints anatomically altered by joint pathology.

Surgical drainage can be done either arthroscopically or with open surgery (3).

Infected joints may be debrided and lavaged using arthroscopic surgery. The installation of antibiotics during open surgery not being recommended (as previously mentioned). Arthroscopy has been shown to have a good followup (3).

Joint Immobilisation and Mobilisation

It has been shown using animal models that prolonged limb immobilisation is not beneficial, but detrimental. Today joint immobilisation is only prescribed in cases where the pain of moving a joint is too incapacitating. It is also necessary in those who undergo surgical drainage. Patients otherwise should be advised to move joints through active and passive ranges of motions within tolerance levels (3,19).

In those too ill to move from bed, passive mobilisation with functional splinting of the joint to preserve its function should be applied (3). It should be noted that manipulation is contra-indicated in joints that are undergoing bacterial arthritis.

Prognosis

The prognosis associated with BA is dependant on the following:

1. The individuals risk factors
2. The joint involved
3. The organism and its virulence
4. Host-microbial interactions
5. Response to treatment
6. Delay in diagnosis (3,14)

According to Christensen et al (1989), a delay of diagnosis by about 10 days is a common situation (33). It is estimated that a delay of over 7 days can endanger the possibility of a successful short term treatment outcome.

BA involvement in infants and neonates, can result in hip problems, which may result in permanent leg length inequality, or predispose the person to hip pathology later in life. If no comorbidities exist, and only one joint is affected, the overall prognosis is good; prognosis of survival and joint function retention is expected. In the elderly patient who is seriously ill with polymicrobial, polyarticular bacterial arthritis with multisystem organ failure, possibly permanent sequelae may occur in the joint, death being a possibility (3).

CONCLUSION

This paper presented the clinical features of BA, and its pathophysiology. The discussion presented an overview of the causes, presentation, and treatment of patients with BA. Chiropractors and osteopaths should consider bacterial infection as an unusual cause of a common presentating complaint ie joint pain.

The review also outlined the associated symptoms and signs of patients presenting with BA, and how they differ from a presentation of a non-infective type of arthritis. The authors hope that this review will increase the awareness of a condition that chiropractors and osteopaths may be overlooking.

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